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Bringmann, L. F.; Eronen, M. I.

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Don't blame the model: Reconsidering the network approach to psychopathology

Laura F. Bringmann

Department of Psychometrics and Statistics, University of Groningen, Netherlands

Interdisciplinary Center of Psychopathology and Emotion regulation (ICPE), University Medical Center Groningen, University of Groningen, Netherlands

Markus I. Eronen

Department of Theory and History of Psychology, University of Groningen, Netherlands

Institute of Philosophy, KU Leuven, Belgium

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Correspondence concerning this article should be addressed to: Laura Bringmann, Faculty of Behavioural and Social Sciences, Department of Psychometrics and Statistics, University of Groningen, Grote Kruisstraat 2/1, 9712 TS, Groningen, The Netherlands. Email: L.F.Bringmann@rug.nl

Abstract

The network approach to psychopathology is becoming increasingly popular. The motivation for this approach is to provide a replacement for the problematic common cause perspective and the associated latent variable model, where symptoms are taken to be mere effects of a common cause (the disorder itself). The idea is that the latent variable model is plausible for medical diseases, but unrealistic for mental disorders, which should rather be conceptualized as networks of directly interacting symptoms. We argue that this rationale for the network approach is misguided. Latent variable (or common cause) models are not inherently problematic, and there is not even a clear boundary where network models end and latent variable (or common cause) models begin. We also argue that focusing on this contrast has led to an unrealistic view of testing and finding support for the network approach, as well as an oversimplified picture of the relationship between medical diseases and mental disorders. As an alternative, we point out more essential contrasts, such as the contrast between dynamic and static modeling approaches, that can provide a better framework for conceptualizing mental disorders. Finally, we discuss several topics and open problems that need to be addressed in order to make the network approach more concrete and to move the field of psychological network research forward.

Keywords: Network; Psychometrics; Latent variable model; Common cause; Psychopathology

Introduction

Networks are everywhere. In the broadest sense, networks can be seen as simplified representations capturing how elements in a system are interconnected. In this light, everything that can be represented as dots (i.e., nodes) with lines (i.e., edges or links) between the dots amounts to a network. Well known networks are the *internet* and the *World Wide Web* (Newman, 2010). Whereas the internet has a clear physical structure (computers are linked by physical cables), the web is a more abstract network, webpages being the nodes and hyperlinks the edges (van Steen, 2010). Furthermore, a prominent *biological* network is the brain, where white matter tracts, or bundles of axons, connect brain regions (Rubinov & Sporns, 2010). Finally, one of the oldest fields in network research is social networks, in which the nodes are individuals, and links between individuals are determined by, for example, their friendship or co-authorship.

Also in psychological research, networks have not been an unfamiliar notion. Techniques like neural networks have been used in cognitive and perceptual psychology, and social networks have played an important role in social psychology for decades (see, for example, Bronfenbrenner, 1986; Mason, Conrey, & Smith, 2007; Posner & Rothbart, 2007; Rubinov & Sporns, 2010). Only recently, however, network research has found its way to psychopathological, emotion, and personality research (Borsboom, Cramer, Schmittmann, Epskamp, & Waldorp, 2011; Bringmann et al., 2013; Costantini et al., 2015; Cramer, van der Sluis, et al., 2012a).

This recent surge in network research in psychology started in psychopathology, where one of the key questions is how mental disorders should be conceptualized. In short, Borsboom, Cramer and colleagues have argued that the dominant common cause perspective, which relies on latent variable models, is problematic in the context of psychopathology (Borsboom, 2008b; Borsboom & Cramer, 2013; Cramer, Waldorp, van der Maas, & Borsboom, 2010b; Cramer, Borsboom, Aggen, & Kendler, 2012; Jones, Heeren, & McNally, 2017; Nuijten, Deserno, Cramer, & Borsboom, 2016). Instead, they suggest that we should conceptualize and study mental disorders as networks of interacting symptoms (Cramer et al., 2010b; Borsboom, 2017b). This compelling reasoning has been taken to heart, and especially in clinical research, an exponential increase in network research is apparent (Fried et al., 2017). Networks representing the symptoms of depressive disorder (Cramer, Borsboom, et al., 2012; Fried & Nesse, 2014; Fried, Nesse, Zivin, Guille, & Sen, 2014; Fried et al., 2015; Robinaugh, LeBlanc, Vuletich, & McNally, 2014; van Borkulo et al., 2015), autism spectrum disorder (Anderson, 2015; Ruzzano, Borsboom, & Geurts, 2015), and post-traumatic stress disorder (McNally, 2012; McNally et al., 2015) have been

introduced, as well as network models of personality traits (Cramer, van der Sluis, et al., 2012a) and diagnostic assessment tools for mental disorders (e.g., BDI, DSM and ICD manuals; Boschloo et al., 2015; Bringmann, Lemmens, Huibers, Borsboom, & Tuerlinckx, 2015; Borsboom et al., 2011; Tio, Epskamp, Noordhof, & Borsboom, 2016), always contrasting this approach to the latent variable or common cause approach. In this paper, we will refer to this perspective as the *network approach*, which has become the standard expression in the literature (e.g., Fried & Cramer, 2017; Borsboom, 2017b).[1]

When the network approach was introduced, several researchers cast doubt on the necessity to counterpose latent variable (or common cause) models and network models (see, e.g., Ashton & Lee, 2012; Danks, Fancsali, Glymour, & Scheines, 2010; Haig & Vertue, 2010; Humphry & McGrane, 2010; Krueger, DeYoung, & Markon, 2010; Markus, 2010; McFarland & Malta, 2010; Molenaar, 2010). This critique, however, has not changed the basic rationale for the network approach, which even in recent articles is still based on the contrast between latent variable and network models (e.g., Hofmann, Curtiss, & McNally, 2016; McNally, 2016; Nuijten et al., 2016; Robinaugh et al., 2014; van der Maas, Kan, Marsman, & Stevenson, 2017).

Our aim in this paper is to take a step back and reconsider the conceptual foundations and the rationale behind the network approach. Focusing on psychopathology, we will critically examine the way common cause and latent variable models have been represented in this literature. We will show that there is no clear boundary where network models end and latent variable (or common cause) models begin. Moreover, we will argue that this is not the essential contrast to emphasize, and that focusing on this contrast has led (among other things) to an unrealistic view of testing and finding support for the network approach, as well as an oversimplified picture of the relationship between medical diseases and mental disorders. As an alternative, we point out more important contrasts, such as the contrast between dynamic and static modeling approaches, that can provide a better framework for conceptualizing mental disorders. Finally, we point out several topics and open problems that need to be addressed in order to make the network approach more concrete and to move the field of psychological network research forward.

[1] As an anonymous referee pointed out, different network-inspired approaches to psychopathology may be possible. Thus, although we use the expression *the* network approach, this does not imply that there is just one possible network approach that can be taken in psychopathology. Moreover, as will become clear later, the network approach itself is very general and consists of many different kinds of models and theoretical ideas.

The network approach

It is a well-observed fact in psychopathology that specific symptoms tend to co-occur (e.g., Lewis, 1934). For example, depressive symptoms are more highly correlated with each other than with symptoms of schizophrenia (Cramer, Borsboom, et al., 2012). Cramer and Borsboom argue that the traditional explanation for this has been to appeal to a latent common cause of the symptoms (Cramer & Borsboom, 2015): symptoms co-occur because they share the same underlying cause or factor. In this picture, the relationship between symptoms such as *insomnia*, *sadness* and *loss of pleasure* is thus not a causal or direct relationship, but merely a correlation that disappears once the real cause (the disorder) is controlled for.

Furthermore, it is argued that psychometrically this corresponds to using a latent variable model where *local independence* is assumed (Borsboom, 2008a; Cramer et al., 2010b; McNally, 2016; Schmittmann et al., 2013). In a latent variable model, the observed variables are assumed to statistically depend on an unobserved latent variable (Markus & Borsboom, 2013a).[2] Formally, this can be expressed as a function connecting the latent variable θ with the expected value of an observed variable x:

$$\mathbf{E}(x|\theta) = \alpha\theta + \varepsilon, \tag{1}$$

α being a regression parameter and ε measurement error (Markus & Borsboom, 2013a, p. 38). The assumption of local independence entails that once you statistically condition on the latent variable (e.g. depression), the dependencies between observed variables (e.g., symptoms) will vanish, rendering the variables or symptoms (locally) independent from each other.

In this latent variable approach, the direct relationships between symptoms are spurious, and symptoms are mere indicators of the underlying disorder (e.g., depression; Cramer et al., 2010b; Reise & Waller, 2009; Schmittmann et al., 2013). Moreover, the unobserved latent variable (e.g., depression) is taken to be the *cause* for the observable symptoms (e.g., *sadness*; Borsboom & Cramer, 2013; Cramer & Borsboom, 2015). As causes need to be distinct from their effects, this would mean that the latent variable approach conceptualizes disorders as entities that are separate from their symptoms (Borsboom & Cramer, 2013; Cramer & Borsboom, 2015).

Based on this, it is argued that this common cause or latent variable approach is suitable for

[2] In this paper, we only discuss *reflective* latent variable models. In *formative* latent variable models, the latent variable is seen as a common effect of the observed variables, and no local independence is assumed. The common cause or latent variable approach that is criticized in the psychological network literature is tied to reflective latent variable models.

medical diseases, but not for mental disorders (Borsboom & Cramer, 2013; Borsboom, 2017b; Cramer & Borsboom, 2015). When you have, for example, human immunodeficiency virus (HIV), common symptoms are *body rash* and *fever* (Paltiel et al., 2005). Having a *body rash* does not seem to be directly causing *fever*. Instead, these symptoms are just indicators of the underlying common cause or latent entity, the medical disease HIV. In mental disorders, however, symptoms seem to directly cause other symptoms: if I am having *sleeping problems*, it causes me to experience more *tiredness*, which in turn can trigger experiencing *sadness*.

Moreover, a person can have HIV without having any symptoms, so HIV and its symptoms are clearly separate from each other. In contrast, it seems unlikely that a mental disorder and its symptoms could be separated in this way (Borsboom & Cramer, 2013). Although you can do a blood test to confirm that somebody has HIV even when there are no symptoms present, it is implausible that you could diagnose somebody with depression without this person having any depressive symptoms. Instead, there seems to be a mereological (part-whole) relation between symptoms and mental disorders: symptoms make up or constitute the disorder, which suggests that depression is not conceptually distinct from its symptoms in the same way that medical diseases are (Borsboom & Cramer, 2013; Cramer et al., 2010b).

Building on this insight, the network approach offers a new conceptualization of mental disorders (Borsboom & Cramer, 2013; Cramer et al., 2010b; Cramer & Borsboom, 2015). In this approach, no underlying common cause for the symptoms is assumed; the co-occurrence of symptoms is rather explained by their direct interactions. Symptoms cause other symptoms, and as the symptom spread continues, it can finally result in a full-blown disorder (Cramer, 2012; Cramer & Borsboom, 2015). Consequently, it is argued that in order to make progress in understanding psychiatric disorders such as depression, symptoms and their interactions should be the focus of research (Fried, 2015; Fried & Nesse, 2015). Thus, instead of a latent variable or common cause approach, a network approach is needed, where psychiatric disorders are conceptualized as dynamic networks of interacting symptoms (Cramer et al., 2010b). These networks representing mental disorders are not only insightful visualizations, but also open a whole new range of novel analyses that arise from the broader field of network research, such as centrality measures, which can highlight symptoms that have a relatively central role in the network (Borsboom & Cramer, 2013; Bringmann et al., 2013).

The purported dichotomy between latent variable (common cause) models and network models has been criticized by several researchers (see, e.g., Haig & Vertue, 2010; Humphry & McGrane, 2010;

Krueger et al., 2010; Markus, 2010; McFarland & Malta, 2010; Molenaar, 2010). First, it has been pointed out that latent variable models and network models are mathematically equivalent. The latent variable model can be transformed into a network model that has an equal number of free parameters and goodness of fit to the data, and vice versa (Molenaar, van Rijn, & Hamaker, 2007; Molenaar, 2010; Epskamp, Maris, Waldorp, & Borsboom, in press; Kruis & Maris, 2016). For example, a one-factor latent variable model can be transformed to a mathematically equivalent network where the edges represent regression relationships between the observed variables and the latent variable is transformed out of the network (Molenaar et al., 2007, p. 189).

Second, network models and latent variable models can be combined (see also Cramer et al., 2016; Epskamp, Maris, et al., in press; Fried & Cramer, 2017; Markus, 2010). This is particularly important for accounting for measurement error. Psychological data tend to be noisy, and also in networks, measurements of symptoms cannot be assumed to be perfect indicators of the attributes that are being measured (i.e., the symptoms). Latent variable models have the advantage of explicitly taking measurement error into account (see also Equation 1; Borsboom, Mellenbergh, & Van Heerden, 2003; Borsboom, 2005; Markus & Borsboom, 2013a; Muthén, 2002). Thus, it is plausible that the network approach also needs latent variable models, for example for each symptom in a network (Asendorpf, 2012; McFarland & Malta, 2010; Markus, 2010).

However, in recent literature on the network approach, these critical points have not been taken to undermine the key insights of the approach. For example, Borsboom (2017a) has argued that the fact that a latent variable model can be transformed to a network model does not mean that the models are theoretically, conceptually or ontologically equivalent (see also Markus, 2002). Additionally, it has been argued that the two models can actually be empirically distinguished with interventions: Whereas the latent variable model (with local independence) will predict that an intervention on one symptom such as sadness will not result in changes in other symptoms, a network model will predict that such an intervention will result in changes propagating through the network (Borsboom, 2017a, see also Cramer, Waldorp, van der Maas, & Borsboom, 2010a; Cramer, Borsboom, et al., 2012). In this way, it is possible to tell the models empirically apart, even if they are statistically equivalent (Borsboom, 2017a).

Finally, the point about combining network models with latent variable models has also been embraced in the psychological network literature, and even statistical models for doing this have been proposed (Epskamp, Rhemtulla, & Borsboom, in press). As combining networks with latent variables, through for example adding a latent variable model for each node in the network, is different from the

approach of using one latent variable to conceptualize the relationship between a mental disorder and its symptoms, Cramer and colleagues consider this to be compatible with the network approach (Cramer et al., 2010a). Thus, up till now the critical points raised against the network approach have not affected its core ideas, and have in fact been incorporated into the approach.

Don't blame the model

In recent network literature, the network approach continues to be justified and introduced based on the contrast between latent variable (or common cause) models and network models (e.g., Borsboom, 2017b; Hofmann et al., 2016; McNally, 2016; Nuijten et al., 2016; Robinaugh et al., 2014; van der Maas et al., 2017). As we saw above, the main argument against latent variable models was that due to their common cause structure they lead to an implausible picture of mental disorders. In this section, we will show that 1) when interpreted causally, latent variable models are not as restrictive as has been put forward, 2) the problems pointed out in the network literature are not due to the models as such, but arise from inserting incompatible variables into a causally interpreted model; and 3) latent variable models do not necessarily have to be interpreted causally.

Let us start with assuming that latent variable models should indeed be interpreted as common cause models, as has been argued by authors advocating the network approach (see also Lahey, Krueger, Rathouz, Waldman, & Zald, 2017; van Bork, Wijsen, & Rhemtulla, in press). As we will explain, common cause models are in fact more complex and flexible than has been assumed in the psychological network literature: even if symptoms of a disorder have a common cause, this does not imply that the symptoms do not directly interact with each other (Danks et al., 2010; Haig & Vertue, 2010; Haslam, 2010; Humphry & McGrane, 2010).

To see this, let us consider a situation where symptom A and symptom B are correlated, and have a common cause C. Now if we condition on C, the correlation between A and B may disappear, rendering A and B locally independent. However, it is also possible that the correlation does not disappear, because A is in fact a direct cause of B. In this case, conditioning on the common cause does not screen off the symptoms from each other, and therefore does not render them locally independent (Danks et al., 2010; Spirtes, Glymour, & Scheines, 2000). Another way of formulating this is as follows: If A and B have a common cause, then that common cause has to explain *some* of the covariance of A and B, but not necessarily *all* of it.

Thus, the existence of a common cause entails local independence only when we already assume

that there are no direct relationships between the symptoms. It is not the case that conditioning on the common cause always "screens off" the symptoms from each other, in contrast to what has been suggested in the psychological network literature (Borsboom, 2008a; Cramer et al., 2010a; Markus & Borsboom, 2013a; McNally, 2016). Perhaps one explanation for the appeal of this assumption is that the reasoning does work the other way around: If we establish that two symptoms are correlated and that there is no direct causal relationship between them, then there are good reasons to believe that there is a (latent) common cause that explains this correlation (Arntzenius, 2010). However, if we start with assuming that symptoms have a common cause, this alone provides no evidence or justification for thinking that they are locally independent, that is, that there is no direct causal relationship between them.

Cramer, van der Sluis, et al. (2012b) have argued that while including direct links between symptoms in a common cause model is possible, it is counterproductive: The more such links you add, the less important the common cause becomes in explaining the covariance between the symptoms. This is true, but only shows that there is no clear dichotomy between networks and common cause models: It is not the case that there are either common causes structures with no direct interactions between symptoms or networks without common causes; instead, there is a whole range of options in between these two extremes. This also means that the idea that there are direct interactions between symptoms is not unique to the network perspective, as it is also consistent with a common cause or latent variable approach.

The second key argument that was raised against latent variable models in the network approach was that they lead to an implausible picture where mental disorders are distinct causes of their own symptoms (see previous section). However, an important thing to note about this problem is that it is not directly linked to the latent variable model. If we assume that the relationship between a mental disorder and its symptoms is constitutive, then using a latent variable model where depression is a latent cause for its symptoms is indeed problematic, but it is equally problematic to use any other kind of causal model (e.g., a DAG) where depression is modeled to cause its symptoms. Thus, the lesson to draw from this is not that latent variable models should be avoided. Rather, the lesson is that any model that includes causal links between constitutively related elements (e.g., the mental disorder and its symptoms) is inconsistent and should not be used.

This shows that it is crucial to include plausible (combinations of) variables in the latent variable models that are studied or tested (see also van Bork, Epskamp, Rhemtulla, Borsboom, & van der Maas,

2017). Importantly, this also has consequences for the common cause hypothesis, which the latent variable model is said to be reflecting (Cramer et al., 2010b). According to this hypothesis, mental disorders (like medical diseases) in the end have a root cause or a common pathogenic pathway that causes the symptoms (Borsboom & Cramer, 2013; Borsboom, 2017b). The causal latent variable models as laid out in the psychological network literature, however, are actually silent on any such etiology of depressive symptoms, as they do not include variables such as brain circuits or neurochemical imbalances as latent variables. Instead, they typically include just *depression* itself as the latent common cause (see, e.g., Borsboom & Cramer, 2013, p. 94). Thus, such latent variable models do not adequately represent the common cause hypothesis described above, and therefore cannot be used to test or falsify it. Instead, to study and test the common cause hypothesis, models that actually include plausible variables representing a root cause or common etiology of the symptoms would be needed.

Finally, all of the considerations against latent variable models in the network literature are based on the common cause structure of these models, and thus require that they be interpreted as causal models. However, in the statistical literature many authors have argued for a non-causal interpretation of latent variable models. For example, a latent variable model can be seen as a parsimonious description of the observed data (Bollen, 2002; Harman, 1960), or as a technique for discovering and efficiently expressing regularities or patterns in the data (Myung, Navarro, & Pitt, 2006). From this perspective, latent variable models are seen as useful but non-causal tools to get insights into the phenomenon of interest (Haig, 2014; Harman, 1960; Jonas & Markon, 2016; Lee, 2012).

Another non-causal way of using latent variable modeling is as an abstraction tool, where the latent variable is literally a common property of the items or indicators of interest (Markus & Borsboom, 2013b; McDonald, 2003). In this approach, the latent variable is seen as conceptually related to the indicators, and not as their cause (McDonald, 2003). Thus, a latent variable such as *depression* can be seen as an "intervening variable" (Hyland, 1981; MacCorquodale & Meehl, 1948) that abstracts over the symptoms loading on this factor, or helps in summarizing the associations between the indicators, without necessarily saying anything about the causal mechanism of depression or how depression comes about.

In light of the above, it is clear that common cause or latent variable models as such are not to blame. There is a broad range of different common cause and latent variable models, and the final interpretation of these models depends on the assumptions imposed, variables selected, and background theories assumed by the researcher. The lack of interactions between the symptoms is not an inherent

feature of common cause models, but an independent assumption, which means that it is perfectly possible to allow for direct interactions between the effect variables in these models. A latent variable model that is causally interpreted is not problematic as such, but *only* when the latent variable represents an entity (e.g., depression) that is not separable from the indicators (e.g., symptoms). And as long as it is interpreted non-causally, the latent variable model can also be used when the factor is *not* distinct from the indicators, that is, not as a model of the (causal) mechanism, but rather as a summary or abstraction tool. Thus, the latent variable model itself is not the source of the problem(s), it should simply be used in a coherent way, by selecting plausible variables as factors and indicators and interpreting the model appropriately.

Reconsidering the rationale for network models

What we have concluded in the previous section suggests that a shift in how we think about the network approach and its rationale is needed. In the network literature, a central reason for why we need network models is that latent variable or common cause models are problematic, but now we have seen that these models do not necessarily have the features that were supposed to make them unfit to study mental disorders. In this section, we will take the next step and argue that the boundaries between common cause and network models, and relatedly between medical diseases and mental disorders, are in general much blurrier than has been assumed in the psychopathological network literature. After this, we will turn to other contrasts that can be more helpful in advancing psychopathological research.

Importantly, as emphasized in the psychological network literature, the scope of network models is very broad (Borsboom & Cramer, 2013). As we pointed out in the introduction, any set of elements (nodes) with connections or relations between them (edges) can be seen as a network. In psychology, networks are usually based on multivariate statistics, where the nodes are the variables of interest (e.g., symptoms), and the edges represent the relationships between the symptoms (e.g., correlation). In the psychological literature, the most popular types of network models are based on (partial) correlations (Epskamp, Rhemtulla, & Borsboom, in press), Ising models (van Borkulo et al., 2014), directed acyclic graphs (McNally, Mair, Mugno, & Riemann, 2017) or vector autoregressive (VAR) models (Bringmann et al., 2013). Additionally, as mentioned above (see section *The network approach*), latent variable networks are also possible, and already appear in the literature (Anandkumar, Hsu, Javanmard, & Kakade, 2013; van der Maas et al., 2006; Epskamp, Maris, et al., in press). In such networks, the nodes are not (only) observed variables or indicators, but latent constructs. Thus, importantly, for something

to be a network, it does not matter whether the nodes are directly observable or latent.

Network models (when causally interpreted, e.g., DAGs) can also include common cause structures. In the simplest sense, whenever a node is a direct cause of two or more other nodes, this amounts to a common cause. Such situations commonly occur in psychopathological networks: For example, *sadness* can be a cause of *suicidal thoughts*, *feelings of guilt* and *anhedonia* (McNally et al., 2017). Moreover, even strict common cause structures where there are no interactions between the effect variables can be seen as (simple) networks, and are in fact known as *star networks* in computer science (Bicsi, 2002). Thus, there seems to be no clear border where network models end and common cause or latent variable models begin.

The same holds for the supposed dichotomy between medical diseases and mental disorders. In the network literature, a stark contrast between these two has been put forward: In medical diseases, there is an underlying biological factor that is a common cause for symptoms that are independent from each other, whereas in mental disorders, causal interactions between symptoms are essential. However, a closer look at the literature on medical diseases shows that this picture is an oversimplification. First, not only symptoms of mental disorders, but also symptoms of medical diseases often directly interact. For example, in lung cancer, there is a direct causal relationship between the symptom coughing and the symptom chest pain, as well as various other complex symptom interactions (Molassiotis, Lowe, Blackhall, & Lorigan, 2011). Second, as network models are defined so broadly, it is not a surprise that they can also be applied to medical diseases. Indeed, "network medicine", where network models are applied to medical diseases, has recently become an emerging subfield of medical science (Barabási, 2011). Thus, medical diseases can exhibit a broad spectrum of causal structures, ranging from simple structures with a single common cause and no symptom interactions to complex and dynamic networks (Haslam, 2010; van Loo, Romeijn, de Jonge, & Schoevers, 2013; Wichers, 2014).[3]

In light of the above, we believe that thinking in terms of contrasts such as "network models vs. latent variable models" or "mental disorders vs. medical diseases" cannot lead to a solid conceptual foundation and rationale for the network approach. Instead, we suggest that the focus should shift to more essential and straightforward contrasts that are already at play in the debate: The network

[3]Medical diseases can also be inseparable from their symptoms in the same way as mental disorders: For example, at present medical diseases such as psoriasis cannot be defined or diagnosed independently of their symptoms (Globe, Bayliss, & Harrison, 2009). In general, the distinction between medical diseases is less clear than has been assumed in the psychological network literature, and the possible connections and differences between the two is a valuable future research topic on its own.

approach puts the focus on parts (or symptoms) instead of wholes (or syndromes); and takes a dynamic instead of static perspective to mental disorders.

The rationale of focusing on symptoms is related to a debate with a long history (Costello, 1992; Mojtabai & Rieder, 1998; Persons, 1986): What kinds of entities should be the primary target of investigation? Should they be the symptoms of a mental disorder, such as depressive symptoms, or the syndrome (depression) as a whole? In the context of the network perspective, this issue has received renewed attention, as the network approach is emphatically focused on the importance of symptoms instead of whole syndromes. This symptom-oriented approach has led to increased understanding of the heterogeneity of symptoms of depression (Fried, 2015), and to explanations of comorbidity that are arguably more plausible than explanations based on syndromes as wholes (Cramer et al., 2010b; Borsboom & Cramer, 2013). This is also in line with the recent Research Domain Criteria (RDoC) initiative, where one of the core ideas is that research should focus on units smaller than whole disorders, and to avoid reifying the diagnostic categories in, for example, the DSM (Kozak & Cuthbert, 2016; Yee, Javitt, & Miller, 2015). Many of the considerations in the network literature can be construed as being against approaches that focus on syndromes as wholes, rather than against latent variable or common cause models as such. From this standpoint, it also makes perfect sense that supporters of the network approach have no objections to using latent variable models for individual symptoms (see above), as the object of study is then still the symptoms instead of the syndrome as a whole.

However, the network approach is not just about looking at the parts or the symptoms, but seems to have a further implicit rationale, namely to bring a dynamic perspective to clinical psychology, thus implying a contrast between static and dynamic approaches (van der Maas et al., 2006; Wichers, Wigman, Bringmann, & de Jonge, 2017).[4] What we mean by "dynamic" here is the idea that symptoms interact with each other and that their interactions change and evolve over time, which is one of the core ideas of the network approach. In the most recent network literature, explicit connections have also been made to the broader field of complex dynamic systems research (Cramer et al., 2016; Borsboom, 2017b).

This kind of dynamic thinking is also at the background in the arguments against latent variable models. The kinds of factor models or latent variable models targeted are static, as they only give a summary of or an abstraction over the covariance structure, and cannot model interaction and changes

[4]We thank Emilio Ferrer and Joseph Gonzales for extensive discussions that led to this insight during our research stay at UC Davis, CA., 2015

over time. However, also in this respect the latent variable model as such should not be seen as the root of the problem, as the important contrast is between dynamic and static approaches, not between network and latent variable models. This can be seen in the fact that latent variable models can also account for dynamics in the form of, for example, a dynamic factor model, which can take time dependencies and thus interactions between variables or symptoms over time into account (Chow, Zu, Shifren, & Zhang, 2011; Ferrer & Nesselroade, 2003; Ferrer, Widaman, Card, Selig, & Little, 2008; Molenaar, 1985; Molenaar, De Gooijer, & Schmitz, 1992). Therefore, we propose that instead of the contrast between latent variable models and network models, or the contrast between medical and mental disorders, the focus in psychopathological modeling should be on other contrasts: Most importantly, static vs. dynamic models and symptom-oriented vs. syndrome-oriented approaches.

Discussion

The network approach is becoming increasingly popular in clinical psychology. However, as we have argued, the focus on the contrast between latent variable and network models has not resulted in a convincing rationale for the network approach, and has drawn the attention away from more essential contrasts. In this section, we will discuss some implications this has, and go through issues that should be addressed in future research.

The first issue concerns the nature of network models. The theoretical idea behind the network perspective is clear: mental disorders should be conceptualized as networks of causally interacting symptoms (Borsboom, 2017b). However, it is not clear what is meant with network models, and thus which models should now be used to study mental disorders. All models that have so far been identified as network models, such as (partial) correlations, DAGs and VAR models, exist outside of the network context, and have already been used in psychological research (see, e.g., Glymour, 2001; Rosmalen, Wenting, Roest, de Jonge, & Bos, 2012). As these so-called network models and the data that they are based on are very diverse, it is unclear what the common denominator of these models would be. Partial correlation analyses are static and typically based on cross-sectional data, whereas VAR models are dynamic in the sense that they explicitly take time into account and are based on time-series data.[5] On the other hand, some otherwise popular models in psychological research, such as path analyses, have not yet been identified as network models, even though path models represent variables (such

[5] Cross-sectional data can also potentially give insights to dynamic processes, but in a more limited way than time-series data (see, e.g., Hamaker, 2012).

as symptoms) with (causal) lines between them and thus at least prima facie look like networks (cf. Hayes, 2013). As no clear criteria for what counts as a network model have been presented in the network approach, it seems to imply that any multivariate model, including path models, can be seen as a network model.

In other words, the network approach is not introducing new kinds of models to psychological research. Instead, the real novelty and distinguishing feature of the network approach, which is important in its own right, seems to be that the existing models (or more precisely, some of their coefficients) are now visualized in an insightful and appealing way. Moreover, the network approach opens up a new toolbox of analyses, such as centrality measures, that can now be applied to these multivariate models. Thus, in our view the advantage of the network approach is not that it provides new modeling solutions per se, but that it suggests new and insightful ways of visualizing existing models and opens up the possibility to calculate network measures on them. Additionally, the network approach has an important heuristic and pragmatic role in guiding researchers and clinicians towards more complex and dynamic ways of thinking about mental disorders.

A further question that deserves more attention is what kinds of variables or nodes should be included in psychopathological networks. The focus has so far been mainly on symptoms and affective states (e.g., Pe et al., 2015; Wigman et al., 2013), but recently it has been suggested that other kinds of nodes, such as social or cognitive factors, should also be included in psychopathological networks (Fried & Cramer, 2017; Jones et al., 2017). In a similar vein, the RDoC initiative emphasizes the importance of incorporating many different units of analysis, such as brain circuits, physiology, and behavior, in addition to just symptoms and affective states (Kozak & Cuthbert, 2016; Yee et al., 2015). However, besides the question of what additional elements should be included in networks, another issue that has been largely disregarded is whether it makes sense to include all symptom variables in a network, as drawing causal lines between them may be problematic on conceptual grounds. In contrast to social networks, where the nodes are people and thus distinct entities, in psychopathological networks the boundaries between the nodes (symptoms) are often fuzzy. For example, does it make sense to infer a causal relationship between variables such as *loss of energy* and *fatigue*, or are they actually overlapping and partly referring to the same thing (see also Fried, Epskamp, Nesse, Tuerlinckx, & Borsboom, 2016; Fried & Cramer, 2017)? In the latter case, these nodes in a network would not be two distinct entities, and representing them as separate nodes with (causal) interactions between them would be problematic. In future research, techniques such as dynamic factor modeling or clustering could help in identifying

and combining overlapping variables (Bulteel, Tuerlinckx, Brose, & Ceulemans, under review).

Additionally, it is important to keep in mind that not all model components are represented in the current network visualizations. Consider, for example, network figures based on the VAR model: They show the effects that variables have on other variables (and themselves) over time, but do not include the mean (and the intercept) or error terms, in contrast to, for example, visualizations of Structural Equation Models (Kline, 2015). This is in line with the aim of the network approach to focus on the interactions between variables instead of the means of each individual variable or symptom. However, this can also be a limitation, as sometimes the changes or effects in mental disorders can only be seen in the means of the individual symptoms of a network. For example, in a study of Snippe and colleagues, the combination of therapy and medication to treat depressive subjects was found to reduce the depressive symptoms on average, but did not have a significant effect on the structure of the symptom network (Snippe et al., 2017). Thus, a challenge for future network research is to find ways of including this kind of important information in network visualizations without losing their clarity and intuitive appeal.

Finally, let us return to the main topic of this paper, namely the contrast between latent variable or common cause models and network models. We have seen that there is no clear border where network models end and latent variable or common cause models begin: even the simplest common cause structures can be seen as networks (e.g., star networks). Nevertheless, it has been suggested in the network literature that future research should focus on developing statistical techniques to compare latent variable models of mental disorders with networks models of mental disorders (Fried & Cramer, 2017, see also Cramer, Borsboom, et al., 2012; Fried et al., 2014). In our view, this is unnecessary or even impossible. In the latent variable models that we find in the network literature, the latent variable is a mental disorder such as depression, which is taken to be the common cause of its symptoms (Borsboom & Cramer, 2013; Fried & Cramer, 2017). These models are then said to be implausible, as depression is (partly) made up of its symptoms, and cannot at the same time cause them (Borsboom & Cramer, 2013). However, what has been overlooked is that this *also* means that there is no need to statistically test such models or to compare them to network models. If a model can be simply ruled out on conceptual grounds, it is not useful (or even possible) to statistically test or compare such an inconsistent model to other models.

As we have suggested in the section *Don't blame the model*, it seems that the arguments against latent variable models presented in the network approach are actually targeting the common cause hypothesis or common cause models. However, it is not yet clear what, according to the network

approach, the common cause hypothesis exactly consists of, or what is meant with common cause models. Is the core idea of the common cause hypothesis simply that there is some root cause (e.g., a neural abnormality, a genetic defect or learned helplessness) to symptoms? In this case, the relevant models are no longer necessarily *latent* variable models: neural abnormalities or genetic defects are arguably more directly observable than symptoms (cf. Borsboom, 2008a). In this picture, it also seems to be entirely irrelevant whether the root causes are latent or not. The key issue is instead whether there *are* root causes to symptoms of mental disorders, not whether those causes are latent or not.[6]

Moreover, this leads to further questions: For example, does the common cause hypothesis *only* refer to models with a single root cause and no interactions between the effects? If yes, it is not clear why only such models should be of interest, as also medical diseases commonly have more complex causal structures, with at least some direct interactions among symptoms. And if interactions among the effects (symptoms) *are* allowed, the contrast between common cause and network models again fades away.

Thus, the terms "network model" or "common cause model" are rather empty in this context. The network approach is not introducing new kinds of models, and network models are understood so generally that also common cause models fall under them. Therefore, testing the network approach has nothing to do with a specific modelling framework per se, but rather amounts to just testing the basic hypothesis that symptoms are (causally) interacting with each other over time. This hypothesis can be tested without the artificial framework of network vs. common cause (or latent variable) models.

Although we have focused here on psychopathological networks, we believe that similar considerations apply to, for example, intelligence, emotion or personality research, where the network approach has also been put forward as an approach that is more plausible than latent variable modeling (Cramer, van der Sluis, et al., 2012a; van der Maas et al., 2006, 2017). Also in these contexts, there is no reason to blame latent variable models, or to rely on the constrictive framework of network vs. common cause models. Thus, our arguments are relevant not only for psychopathology, but for all fields of psychology where the network approach can be potentially applied. We hope that the insights developed in this paper will contribute to advancing and clarifying the rapidly expanding field of network research in

[6]Often it seems that the main aim of the network approach is in fact to argue against a reductionist or biology-based view of mental disorders (see especially Borsboom, Cramer, & Kalis, in press). We agree that such reductionism is implausible (see also Miller, 2010), but do not think that the contrast between network and common cause models is a helpful way of framing this debate: On the one hand, common causes can be irreducibly psychological, and on the other, networks can also contain biological variables or be reducible to biological mechanisms.

psychopathology and psychology more generally.

References

Anandkumar, A., Hsu, D., Javanmard, A., & Kakade, S. (2013). Learning linear bayesian networks with latent variables. In S. Dasgupta & D. McAllester (Eds.), *Proceedings of the 30th international conference on machine learning* (pp. 249–257).

Anderson, G. M. (2015). Autism biomarkers: challenges, pitfalls and possibilities. *Journal of Autism and Developmental Disorders*, *45*(4), 1103–1113.

Arntzenius, F. (2010). Reichenbach's common cause principle. In E. N. Zalta (Ed.), *The stanford encyclopedia of philosophy* (Fall 2010 ed.). Metaphysics Research Lab, Stanford University. `https://plato.stanford.edu/archives/fall2010/entries/physics-Rpcc/`.

Asendorpf, J. B. (2012). What do the items and their associations refer to in a network approach to personality? *European Journal of Personality*, *26*(4), 432–433.

Ashton, M. C., & Lee, K. (2012). On models of personality structure. *European Journal of Personality*, *26*(4), 433–434.

Barabási, A.-L. (2011). The network takeover. *Nature Physics*, *8*(1), 14-16.

Bicsi, B. (2002). *Network design basics for cabling professionals.* New York: McGraw-Hill.

Bollen, K. (2002). Latent variables in psychology and the social sciences. *Annual Review of Psychology*, *53*(1), 605–634.

Borsboom, D. (2005). *Measuring the mind: Conceptual issues in contemporary psychometrics.* Cambridge: Cambridge University Press.

Borsboom, D. (2008a). Latent variable theory. *Measurement: Interdisciplinarity Research and Perspectives*, *6*, 25–53.

Borsboom, D. (2008b). Psychometric perspectives on diagnostic systems. *Journal of clinical psychology*, *64*(9), 1089–1108.

Borsboom, D. (2017a). *The meaning of model equivalence: Network models, latent variables, and the theoretical space in between* (Blog post No. January 27). http://psych-networks.com/meaning-model-equivalence-network-models-latent-variables-theoretical-space/.

Borsboom, D. (2017b). A network theory of mental disorders. *World Psychiatry*, *16*(1), 5–13.

Borsboom, D., & Cramer, A. (2013). Network analysis: An integrative approach to the structure of psychopathology. *Annual Review of Clinical Psychology*, *9*, 91–121.

Borsboom, D., Cramer, A., & Kalis, A. (in press). Brain disorders? not really why network structures block reductionism in psychopathology research. *Behavioral and Brain Sciences*, 1–54. doi: https://doi.org/10.1017/S0140525X17002266

Borsboom, D., Cramer, A., Schmittmann, V. D., Epskamp, S., & Waldorp, L. J. (2011). The small world of psychopathology. *PloS ONE*, *6*(11), e27407.

Borsboom, D., Mellenbergh, G. J., & Van Heerden, J. (2003). The theoretical status of latent variables. *Psychological Review*, *110*(2), 203–219.

Boschloo, L., van Borkulo, C. D., Rhemtulla, M., Keyes, K. M., Borsboom, D., & Schoevers, R. A. (2015). The network structure of symptoms of the diagnostic and statistical manual of mental disorders. *PloS ONE*, *10*(9), e0137621.

Bringmann, L. F., Lemmens, L. H., Huibers, M. J., Borsboom, D., & Tuerlinckx, F. (2015). Revealing the dynamic network structure of the Beck Depression Inventory-II. *Psychological Medicine*, *45*(4), 747–757.

Bringmann, L. F., Vissers, N., Wichers, M., Geschwind, N., Kuppens, P., Peeters, F., ... Tuerlinckx, F. (2013). A network approach to psychopathology: New insights into clinical longitudinal data. *PLoS ONE*, *8*(4), e60188.

Bronfenbrenner, U. (1986). Ecology of the family as a context for human development: Research perspectives. *Developmental Psychology*, *22*(6), 723–742.

Bulteel, K., Tuerlinckx, F., Brose, A., & Ceulemans, E. (under review). Improved insight into and prediction of network dynamics by combining var and dimension reduction.

Chow, S.-M., Zu, J., Shifren, K., & Zhang, G. (2011). Dynamic factor analysis models with time-varying parameters. *Multivariate Behavioral Research*, *46*(2), 303–339.

Costantini, G., Epskamp, S., Borsboom, D., Perugini, M., Mõttus, R., Waldorp, L. J., & Cramer, A. O. (2015). State of the aRt personality research: A tutorial on network analysis of personality data in R. *Journal of Research in Personality*, *54*, 13–29.

Costello, C. G. (1992). Research on symptoms versus research on syndromes. Arguments in favour of allocating more research time to the study of symptoms. *The British Journal of Psychiatry*, *160*(3), 304–308.

Cramer, A. (2012). Why the item "23 + 1" is not in a depression questionnaire: Validity from a network perspective. *Measurement: Interdisciplinary Research and Perspective*, *10*(1-2), 50–54.

Cramer, A., & Borsboom, D. (2015). Problems attract problems: a network perspective on mental disorders. *Emerging Trends in the Social and Behavioral Sciences: An Interdisciplinary, Searchable, and Linkable Resource*, 115. doi: 10.1002/9781118900772.etrds0264

Cramer, A., Borsboom, D., Aggen, S., & Kendler, K. (2012). The pathoplasticity of dysphoric episodes: Differential impact of stressful life events on the pattern of depressive symptom inter-correlations. *Psychological Medicine*, *42*, 957–965.

Cramer, A., van Borkulo, C., Giltay, E., van der Maas, H., Kendler, K., Scheffer, M., & Borsboom, D. (2016). Major depression as a complex dynamical system. *PLoS ONE*, *11*, e0167490.

Cramer, A., van der Sluis, S., Noordhof, A., Wichers, M., Geschwind, N., Aggen, S. H., ... Borsboom, D. (2012a). Dimensions of normal personality as networks in search of equilibrium: You can't like parties if you don't like people. *European Journal of Personality*, *26*(4), 414–431.

Cramer, A., van der Sluis, S., Noordhof, A., Wichers, M., Geschwind, N., Aggen, S. H., ... Borsboom, D. (2012b). Measurable like temperature or mereological like flocking? On the nature of personality traits. *European Journal of Personality*, *26*(4), 451–459.

Cramer, A., Waldorp, L. J., van der Maas, H. L., & Borsboom, D. (2010a). Authors' response. Complex realities require complex theories: Refining and extending the network approach to mental disorders. *Behavioral and Brain Sciences*, *33*(2-3), 178193.

Cramer, A., Waldorp, L. J., van der Maas, H. L., & Borsboom, D. (2010b). Comorbidity: A network perspective. *Behavioral and Brain Sciences*, *33*(2-3), 137–150.

Danks, D., Fancsali, S., Glymour, C., & Scheines, R. (2010). Comorbid science? *Behavioral and Brain Sciences*, *33*(2-3), 153–155.

Epskamp, S., Maris, G., Waldorp, L. J., & Borsboom, D. (in press). Network psychometrics. In P. Irwing, D. Hughes, & T. Booth (Eds.), *Handbook of Psychometrics.* New York, NY: John Wiley & Sons.

Epskamp, S., Rhemtulla, M., & Borsboom, D. (in press). Generalized network psychometrics: Combining network and latent variable models. *Psychometrika*.

Ferrer, E., & Nesselroade, J. R. (2003). Modeling affective processes in dyadic relations via dynamic factor analysis. *Emotion*, *3*(4), 344-360.

Ferrer, E., Widaman, K. F., Card, N., Selig, J., & Little, T. (2008). Dynamic factor analysis of dyadic affective processes with inter-group differences. In N. Card, J. Selig, & T. Little (Eds.), *Modeling dyadic and interdependent data in the developmental and behavioral sciences* (pp. 107–137). Hillsdale, NJ: Psychology Press.

Fried, E. I. (2015). Problematic assumptions have slowed down depression research: Why symptoms, not syndromes are the way forward. *Frontiers in Psychology*, *6*, 1–11.

Fried, E. I., Bockting, C., Arjadi, R., Borsboom, D., Amshoff, M., Cramer, A. O., ... Stroebe, M. (2015). From loss to loneliness: The relationship between bereavement and depressive symptoms. *Journal of Abnormal Psychology*, *124*(2), 256–265.

Fried, E. I., & Cramer, A. (2017). Moving forward: challenges and directions for psychopathological network theory and methodology. *Perspectives on Psychological Science*, *12*(6), 999–1020.

Fried, E. I., Epskamp, S., Nesse, R. M., Tuerlinckx, F., & Borsboom, D. (2016). What are 'good' depression symptoms? Comparing the centrality of DSM and non-DSM symptoms of depression in a network analysis. *Journal of Affective Disorders*, *189*, 314–320.

Fried, E. I., & Nesse, R. M. (2014). The impact of individual depressive symptoms on impairment of psychosocial functioning. *PLoS ONE*, *9*(2), e90311.

Fried, E. I., & Nesse, R. M. (2015). Depression sum-scores dont add up: why analyzing specific depression symptoms is essential. *BMC Medicine*, *13*, 1–11.

Fried, E. I., Nesse, R. M., Zivin, K., Guille, C., & Sen, S. (2014). Depression is more than the sum score of its parts: Individual DSM symptoms have different risk factors. *Psychological Medicine*, *44*(10), 2067–2076.

Fried, E. I., van Borkulo, C. D., Cramer, A. O., Boschloo, L., Schoevers, R. A., & Borsboom, D. (2017). Mental disorders as networks of problems: a review of recent insights. *Social Psychiatry and Psychiatric Epidemiology*, *52*(1). doi: 10.1007/s00127-016-1319-z

Globe, D., Bayliss, M. S., & Harrison, D. J. (2009). The impact of itch symptoms in psoriasis: results from physician interviews and patient focus groups. *Health and quality of life outcomes*, *7*(1), 62.

Glymour, C. N. (2001). *The mind's arrows: Bayes nets and graphical causal models in psychology.* MIT press.

Haig, B. D. (2014). *Investigating the psychological world: Scientific method in the behavioral sciences.* Cambridge, MA: MIT press.

Haig, B. D., & Vertue, F. M. (2010). Extending the network perspective on comorbidity. *Behavioral and Brain Sciences*, *33*(2-3), 158–158.

Hamaker, E. L. (2012). Why researchers should think within-person: A paradigmatic rationale. In M. R. Mehl & T. S. Conner (Eds.), *Handbook of research methods for studying daily life* (pp. 43–61). New York, NY: Guilford.

Harman, H. (1960). *Modern factor analysis.* Chicago: University of Chicago Press.

Haslam, N. (2010). Symptom networks and psychiatric categories. *Behavioral and Brain Sciences*, *33*(2-3), 158–159.

Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach.* New York: Guilford Press.

Hofmann, S. G., Curtiss, J., & McNally, R. J. (2016). A complex network perspective on clinical science. *Perspectives on Psychological Science*, *11*(5), 597–605.

Humphry, S. M., & McGrane, J. A. (2010). Is there a contradiction between the network and latent variable perspectives? *Behavioral and Brain Sciences*, *33*(2-3), 160–161.

Hyland, M. (1981). Hypothetical constructs and intervening variables. In *Introduction to theoretical psychology* (pp. 32–41). London: MacMillan.

Jonas, K. G., & Markon, K. E. (2016). A descriptivist approach to trait conceptualization and inference. *Psychological Review*, *123*(1), 90–96.

Jones, P. J., Heeren, A., & McNally, R. J. (2017). Commentary: A network theory of mental disorders.

Frontiers in psychology, *8*, 1305.

Kline, R. B. (2015). *Principles and practice of structural equation modeling*. New York, NY: Guilford.

Kozak, M. J., & Cuthbert, B. N. (2016). The NIMH research domain criteria initiative: background, issues, and pragmatics. *Psychophysiology*, *53*(3), 286–297.

Krueger, R. F., DeYoung, C. G., & Markon, K. E. (2010). Toward scientifically useful quantitative models of psychopathology: The importance of a comparative approach. *Behavioral and Brain Sciences*, *33*(2-3), 163–164.

Kruis, J., & Maris, G. (2016). Three representations of the Ising model. *Scientific Reports*, *6*. doi: http://dx.doi.org/10.1038/srep34175

Lahey, B. B., Krueger, R. F., Rathouz, P. J., Waldman, I. D., & Zald, D. H. (2017). A hierarchical causal taxonomy of psychopathology across the life span. *Psychological bulletin*, *143*(2), 142.

Lee, J. J. (2012). Common factors and causal networks. *European Journal of Personality*, *26*(4), 441–442.

Lewis, A. J. (1934). Melancholia: a clinical study of depressive states. *The British Journal of Psychiatry*, *80*, 277–378.

MacCorquodale, K., & Meehl, P. E. (1948). On a distinction between hypothetical constructs and intervening variables. *Psychological review*, *55*(2), 95–107.

Markus, K. A. (2002). Statistical equivalence, semantic equivalence, eliminative induction, and the Raykov-Marcoulides proof of infinite equivalence. *Structural Equation Modeling*, *9*(4), 503–522.

Markus, K. A. (2010). Questions about networks, measurement, and causation. *Behavioral and Brain Sciences*, *33*(2-3), 164–165.

Markus, K. A., & Borsboom, D. (2013a). *Frontiers of test validity theory: Measurement, causation, and meaning*. London: Routledge.

Markus, K. A., & Borsboom, D. (2013b). Reflective measurement models, behavior domains, and common causes. *New Ideas in Psychology*, *31*(1), 54–64.

Mason, W. A., Conrey, F. R., & Smith, E. R. (2007). Situating social influence processes: Dynamic, multidirectional flows of influence within social networks. *Personality and Social Psychology Review*, *11*(3), 279–300.

McDonald, R. P. (2003). Behavior domains in theory and in practice. *Alberta Journal of Educational Research*, *49*, 212–230.

McFarland, D. J., & Malta, L. S. (2010). Symptoms as latent variables. *Behavioral and Brain Sciences*, *33*(2-3), 165–166.

McNally, R. J. (2012). The ontology of posttraumatic stress disorder: Natural kind, social construction, or causal system? *Clinical Psychology: Science and Practice*, *19*(3), 220–228.

McNally, R. J. (2016). Can network analysis transform psychopathology? *Behaviour Research and Therapy*, *86*, 95–104.

McNally, R. J., Mair, P., Mugno, B., & Riemann, B. (2017). Co-morbid obsessive–compulsive disorder and depression: a bayesian network approach. *Psychological medicine*, *47*(7), 1204–1214.

McNally, R. J., Robinaugh, D. J., Wu, G. W., Wang, L., Deserno, M. K., & Borsboom, D. (2015). Mental disorders as causal systems: A network approach to posttraumatic stress disorder. *Clinical Psychological Science*, *3*(6), 836–849.

Miller, G. A. (2010). Mistreating psychology in the decades of the brain. *Perspectives on Psychological Science*, *5*(6), 716–743.

Mojtabai, R., & Rieder, R. O. (1998). Limitations of the symptom-oriented approach to psychiatric research. *The British Journal of Psychiatry*, *173*(3), 198–202.

Molassiotis, A., Lowe, M., Blackhall, F., & Lorigan, P. (2011). A qualitative exploration of a respiratory distress symptom cluster in lung cancer: cough, breathlessness and fatigue. *Lung Cancer*, *71*(1), 94–102.

Molenaar, P. C. M. (1985). A dynamic factor model for the analysis of multivariate time series. *Psychometrika*, *50*(2), 181–202.

Molenaar, P. C. M. (2010). Latent variable models are network models. *Behavioral and Brain Sciences*, *33*(2-3), 166.

Molenaar, P. C. M., De Gooijer, J. G., & Schmitz, B. (1992). Dynamic factor analysis of nonstationary multivariate time series. *Psychometrika*, *57*(3), 333–349.

Molenaar, P. C. M., van Rijn, P., & Hamaker, E. L. (2007). A new class of sem model equivalences and its implications. In S. M. Boker & J. M. Wenger (Eds.), *Data analytic techniques for dynamical systems* (pp. 189–211). Hillsdale, NJ: Erlbaum.

Muthén, B. O. (2002). Beyond SEM: General latent variable modeling. *Behaviormetrika*, *29*(1), 81–117.

Myung, J. I., Navarro, D. J., & Pitt, M. A. (2006). Model selection by normalized maximum likelihood. *Journal of Mathematical Psychology*, *50*(2), 167–179.

Newman, M. (2010). *Networks: An introduction*. Oxford: Oxford University Press.

Nuijten, M., Deserno, M., Cramer, A., & Borsboom, D. (2016). Mental disorders as complex networks: An introduction and overview of a network approach to psychopathology. *Clinical Neuropsychiatry*, *13*, 68–76.

Paltiel, A. D., Weinstein, M. C., Kimmel, A. D., Seage III, G. R., Losina, E., Zhang, H., ... Walensky, R. P. (2005). Expanded screening for HIV in the United States: An analysis of cost-effectiveness. *New England Journal of Medicine*, *352*(6), 586–595.

Pe, M. L., Kircanski, K., Thompson, R. J., Bringmann, L. F., Tuerlinckx, F., Mestdagh, M., ... Gotlib, I. H.

(2015). Emotion-network density in major depressive disorder. *Clinical Psychological Science*, *3*(2), 292–300.

Persons, J. B. (1986). The advantages of studying psychological phenomena rather than psychiatric diagnoses. *American Psychologist*, *41*(11), 12521260.

Posner, M. I., & Rothbart, M. K. (2007). Research on attention networks as a model for the integration of psychological science. *Annual Review of Psychology*, *58*, 1–23.

Reise, S. P., & Waller, N. G. (2009). Item response theory and clinical measurement. *Annual review of clinical psychology*, *5*, 27–48.

Robinaugh, D. J., LeBlanc, N. J., Vuletich, H. A., & McNally, R. J. (2014). Network analysis of persistent complex bereavement disorder in conjugally bereaved adults. *Journal of Abnormal Psychology*, *123*(3), 510-522.

Rosmalen, J. G., Wenting, A. M., Roest, A. M., de Jonge, P., & Bos, E. H. (2012). Revealing causal heterogeneity using time series analysis of ambulatory assessments: Application to the association between depression and physical activity after myocardial infarction. *Psychosomatic Medicine*, *74*(4), 377–386.

Rubinov, M., & Sporns, O. (2010). Complex network measures of brain connectivity: Uses and interpretations. *NeuroImage*, *52*(3), 1059–1069.

Ruzzano, L., Borsboom, D., & Geurts, H. M. (2015). Repetitive behaviors in autism and obsessive–compulsive disorder: New perspectives from a network analysis. *Journal of Autism and Developmental Disorders*, *45*(1), 192–202.

Schmittmann, V. D., Cramer, A., Waldorp, L. J., Epskamp, S., Kievit, R. A., & Borsboom, D. (2013). Deconstructing the construct: A network perspective on psychological phenomena. *New Ideas in Psychology*, *31*(1), 43–53.

Snippe, E., Viechtbauer, W., Geschwind, N., Klippel, A., De Jonge, P., & Wichers, M. (2017). The impact of treatments for depression on the dynamic network structure of mental states: Two randomized controlled trials. *Scientific Reports*, *7*. doi: 10.1038/srep46523

Spirtes, P., Glymour, C. N., & Scheines, R. (2000). *Causation, prediction, and search*. Cambridge, MA: MIT press.

Tio, P., Epskamp, S., Noordhof, A., & Borsboom, D. (2016). Mapping the manuals of madness: Comparing the ICD-10 and DSM-IV-TR using a network approach. *International Journal of Methods in Psychiatric Research*, *25*(4), 267–276.

van Bork, R., Epskamp, S., Rhemtulla, M., Borsboom, D., & van der Maas, H. (2017). What is the p-factor of psychopathology? Some risks of general factor modeling. *Theory and Psychology*, *27*(6), 759–773.

van Bork, R., Wijsen, L. D., & Rhemtulla, M. (in press). Toward a causal interpretation of the reflective

measurement model. *Disputatio*.

van Borkulo, C. D., Borsboom, D., Epskamp, S., Blanken, T. F., Boschloo, L., Schoevers, R. A., & Waldorp, L. J. (2014). A new method for constructing networks from binary data. *Scientific reports*, *4*. doi: 10.1038/srep05918

van Borkulo, C. D., Boschloo, L., Borsboom, D., Penninx, B. W., Waldorp, L. J., & Schoevers, R. A. (2015). Association of symptom network structure with the course of longitudinal depression. *JAMA Psychiatry*, *72*(12), 1219–1226.

van der Maas, H., Dolan, C. V., Grasman, R. P., Wicherts, J. M., Huizenga, H. M., & Raijmakers, M. E. (2006). A dynamical model of general intelligence: The positive manifold of intelligence by mutualism. *Psychological Review*, *113*(4), 842–861.

van der Maas, H., Kan, K.-J., Marsman, M., & Stevenson, C. E. (2017). Network models for cognitive development and intelligence. *Journal of Intelligence*, *5*(2). doi: 10.3390/jintelligence5020016

van Loo, H. M., Romeijn, J.-W., de Jonge, P., & Schoevers, R. A. (2013). Psychiatric comorbidity and causal disease models. *Preventive Medicine*, *57*(6), 748–752.

van Steen, M. (2010). *Graph theory and complex networks: An introduction.* Amsterdam: Maarten van Steen.

Wichers, M. (2014). The dynamic nature of depression: a new micro-level perspective of mental disorder that meets current challenges. *Psychological Medicine*, *44*(7), 1349–1360.

Wichers, M., Wigman, J., Bringmann, L., & de Jonge, P. (2017). Mental disorders as networks: some cautionary reflections on a promising approach. *Social Psychiatry and Psychiatric Epidemiology*, *52*(2), 143–145.

Wigman, J., van Os, J., Thiery, E., Derom, C., Collip, D., Jacobs, N., & Wichers, M. (2013). Psychiatric diagnosis revisited: towards a system of staging and profiling combining nomothetic and idiographic parameters of momentary mental states. *PLoS One*, *8*(3), e59559.

Yee, C. M., Javitt, D. C., & Miller, G. A. (2015). Replacing DSM categorical analyses with dimensional analyses in psychiatry research: the research domain criteria initiative. *JAMA psychiatry*, *72*(12), 1159–1160.